ACCESSION NUMBER: 90386068 MEDLINE
DOCUMENT NUMBER: 90386068 PubMed ID: 2206035
TITLE: **Lovastatin** and **simvastatin** **prevention** studies.
AUTHOR: Jones P H
CORPORATE SOURCE: Baylor College of Medicine, Houston, Texas 77030.
SOURCE: AMERICAN JOURNAL OF CARDIOLOGY, (1990 Sep 18) 66 (8) 39B-43B. Ref: 20
Journal code: 0207277. ISSN: 0002-9149.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
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LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199010
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AB There is substantial evidence that drug treatment of hypercholesterolemia in patients without known coronary artery disease (CAD) can reduce fatal and nonfatal CAD events. Two trials, the Lipid Research Clinics Coronary Primary **Prevention** Trial and the Helsinki Heart Study used cholestyramine and gemfibrozil, respectively, to alter lipoprotein levels; their demonstrated efficacy and safety have led to their widespread use in hyperlipidemic patients. Recently, a new class of hypolipidemic agents has been shown to be extremely effective in lowering total and low-density lipoprotein cholesterol levels. These drugs, including **lovastatin** and **simvastatin**, competitively inhibit 3-hydroxy-3-methylglutaryl coenzyme A reductase, the rate-limiting enzyme of intracellular cholesterol synthesis. Results of safety and efficacy studies suggest that they may be valuable first-line treatment options for high-risk hypercholesterolemic patients. Two long-term clinical trials are planned with **lovastatin** and **simvastatin**. In the United States, **lovastatin** will be used in a double-blind, placebo-controlled, primary **prevention** trial involving 8,000 patients without clinical evidence of CAD, slight to moderate elevations of total cholesterol, and low- and high-density lipoprotein cholesterol to establish whether 5 years of treatment will decrease the rate of fatal CAD or nonfatal myocardial infarction. A Scandinavian study of 4,000 patients with ischemic heart disease and hypercholesterolemia will determine if **simvastatin** will improve total survival and reduce fatal or nonfatal myocardial infarction and sudden death for at least 3 years. These study designs will be discussed and compared with other studies, and the expected impact on CAD event rates presented.

ACCESSION NUMBER: 90358133 MEDLINE
DOCUMENT NUMBER: 90358133 PubMed ID: 1975155
TITLE: Pharmacologic management of ischemic heart disease with beta-blockers and calcium channel blockers.
AUTHOR: Pearle D L
CORPORATE SOURCE: Division of Cardiology, Georgetown University Medical Center, Washington, DC 20007.
SOURCE: AMERICAN HEART JOURNAL, (1990 Sep) 120 (3) 739-42; discussion 743-5.
Journal code: 0370465. ISSN: 0002-8703.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199009
ENTRY DATE: Entered STN: 19901026
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Entered Medline: 19900927
AB In myocardial ischemia beta-blockers reduce myocardial oxygen demand, improve flow toward ischemic regions, and have mild antiplatelet and antiarrhythmic effects. These agents are effective in chronic stable angina and unstable angina. In chronic myocardial ischemia, the beta-blockers **timolol**, **metoprolol**, **atenolol**, and **propranolol** have **cardioprotective** effects, reducing overall mortality and the incidence of recurrent myocardial infarction. Calcium channel blockers, which reduce myocardial oxygen demand and improve oxygen supply, are effective in the treatment of chronic stable angina, vasospastic angina, and unstable angina. Although calcium channel blockers generally have no effect or adverse effects when used as primary therapy for acute myocardial infarction, diltiazem (when used concomitantly with nitrates or beta-blockers) has been shown to reduce the incidence of reinfarction in patients after non-Q wave myocardial infarction.

ACCESSION NUMBER: 1999010017 MEDLINE
DOCUMENT NUMBER: 99010017 PubMed ID: 9793602
TITLE: Drug and environmental factors associated with adverse pregnancy outcomes. Part III: Folic acid: pharmacology, therapeutic recommendations, and economics.
AUTHOR: Lewis D P; Van Dyke D C; Stumbo P J; Berg M J
CORPORATE SOURCE: College of Pharmacy, University of Iowa, Iowa City, IA 52242, USA.
CONTRACT NUMBER: MO1-5500059
SOURCE: ANNALS OF PHARMACOTHERAPY, (1998 Oct) 32 (10) 1087-95.
Ref: 119
Journal code: 9203131. ISSN: 1060-0280.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981216
AB OBJECTIVE: To review **folic acid**'s mechanism of action, adverse effects, therapeutic recommendations, compliance, and cost. DATA SOURCES: A MEDLINE search was conducted through December 1997. Additional sources were obtained from Current Contents and citations from the references obtained. Search terms included **folate**, **folic acid**, neural tube defect, homocysteine, and methylenetetrahydrofolate reductase. STUDY SELECTION: Animal and human studies examining the effects of **folate** were reviewed. DATA EXTRACTION: Data collected included mechanism of action, safety issues, dosing recommendations, compliance with recommendations, and economics. DATA SYNTHESIS: **Folic acid** decreases neural tube defect risk through an effect on methionine-homocysteine metabolism. In addition, increased **folate** intake may reduce **cardiovascular** morbidity and mortality. Since toxicity is minimal, everyone can potentially benefit from increased **folate** consumption. To help achieve this, the Food and Drug Administration has mandated that cereal grain be fortified with 140 micrograms of **folic acid** per 100 g of grain, which will add approximately 0.1 mg of **folate** to the average diet. Studies recommend supplementing with 0.2 mg to promote optimal homocysteine concentrations and for **preventing** neural tube defects. CONCLUSIONS: Despite fortification, most women will still receive less **folate** than the 0.4 mg/d recommended by the Public Health Service. All population groups would benefit from increased **folate** intake. Current studies indicate 200 micrograms/d may be the minimum effective amount of fortification needed for normalizing homocysteine concentrations and **preventing** a significant number of neural tube defects; thus, a higher level of food fortification may be warranted.

ACCESSION NUMBER: 1999010793 MEDLINE
DOCUMENT NUMBER: 99010793 PubMed ID: 9796769
TITLE: Homocysteine and cardiovascular disease.
AUTHOR: Abby S L; Harris I M; Harris K M
CORPORATE SOURCE: Deaconess Family Medicine Residency Program, St. Louis College of Pharmacy, MO, USA.
SOURCE: JOURNAL OF THE AMERICAN BOARD OF FAMILY PRACTICE, (1998 Sep-Oct) 11 (5) 391-8. Ref: 43
Journal code: 8807505. ISSN: 0893-8652.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
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AB After a thorough review of the available literature, it appears that hyperhomocysteinemia is an independent risk factor for CHD. Furthermore, **folic acid** has been shown to reduce homocysteine concentration. Nevertheless, CHD is a multifactorial process, and many risk factors play a role in its pathogenesis. Several unanswered questions remain regarding the role of **folic acid** supplementation in hyperhomocysteinemia (Table 3). The absolute homocysteine concentration at which **cardiovascular** risk increases is not certain, and the magnitude of homocysteine-lowering needed to **prevent** events is unknown. Consequently, the number needed to treat cannot be calculated for **folic acid** supplements. Based on these data, the populations in whom to evaluate a homocysteine concentration have yet to be described. Because the POEMs are not yet available, it is unknown whether supplemental **folic acid** to lower homocysteine concentration will reduce CHD morbidity and mortality. It will take several years before any randomized, controlled trials are done, and primary **prevention** trials will need to be of very long duration to show any change in outcomes. Widespread use of **folic acid** supplementation has been recommended, however, and the need for clinical outcomes might be precluded. Even in the absence of outcome data, the potential benefits of using **folic acid** appear to outweigh any risks. A diet high in **folic acid** should be encouraged in everyone (Table 4). The FDA-mandated **folic acid** fortification of enriched grain products is most likely insufficient to lower homocysteine concentrations meaningfully, and a daily multivitamin that contains 400 microg of **folic acid** should be considered for patients who have documented CHD (especially when other risk factors are absent or in patients with premature atherosclerosis) and men and women who have **cardiovascular** risk factors, in addition to women of childbearing potential. **Folic acid** supplementation in the form of a multivitamin once daily is safe and inexpensive and might **prevent** the development and progression of CH